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Case report

What may be happen after an organophosphate exposure: Acute myocardial infarction?

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ABSTRACT

The increase in accidental organophosphate poisoning as well as the rise in the number of cases of suicide attempts with organophosphate compounds is due to primarily to the widespread use of these compounds in agriculture. Organophosphates are anti-acetycholinesterase agents and their toxicity affects many organs, including the pancreas, liver and heart. Cardiac complications often accompany poisoning with these compounds and may be serious and often fatal. However, little is known about the myocardial infarction risk associated with exposure to pesticides. Herein, a rare case of acute myocardial infarction due to acute exposure to organophosphate compound is documented with electrocardiogram, enzyme and clinical characteristics in this report.

1. Introduction

Organophosphates (OPs) are used as insecticides in agricultural and domestic settings throughout the world.[1] This easy availability of the compounds has resulted in a gradual increase in accidental and suicidal poisoning mainly in developing countries.[2] We report a rare case of 52-year-old man who suffered an inferior myocardial infarction following exposure to OPs compound.

2. Case report

A 52- year-old farmer man was admitted to emergency department with severe chest pain. It was started half an hour after ingestion of an unknown quantity of parathion with accidentally during the course of work. Duration of chest pain was approximately 1 h prior to coming hospital. His other chief complaints were dizziness, myalgia, vomiting, increased salivation and diaphoresis. He didn't have any specific neurological symptoms, muscle weakness. His blood pressure was 110/70 mm Hg and his heart rate was 80 beats/min. He was a smoker (13 cigarettes/day) for thirty years and did not describe any exertional symptoms or any significant medical history and regular medications. The patient's laboratory findings, demonstrated hyperglycemia, leucocytosis, mild anemia and hyponatremia. Cardiac markers were elevated at time of admission (Table 1). Serum cholinesterase level couldn't measured because of insufficient laboratory conditions. The electrocardiogram showed 1–2-mm ST-segment elevation in DII, DIII and AVF derivations accompanied by 1–2 mm ST horizontal depression in DI-AVL leads (Fig. 1). The diagnosis was acute inferior myocardial infarction. Antiplatelet agents with aspirin 300 mg, clopidogrel 600 mg, and an antithrombin agent with heparin 10.000 unit bolus were used prior to coronary angiography. After this treatment a coronary angiography was done immediately, door to balloon time was roughly half an hour. Coronary angiography demonstrated 90% stenosis of the right coronary artery which was succesfully treated concurrently with deployment of one bare metal stent and he was discharged two day after with medical therapy and without any adverse events.

3. Discussion

Cardiac complications often accompany poisoning with organophosphates.[3] These may be serious and often fatal, being represented by cardiac arrhythmias, electrocardiographic abnormalities and conduction defects, as well as myocardial infarction, a rarely reported complication of acute pesticide poisoning.[4]

The mechanism by which organophosphate poisoning induces cardiac toxicity has not been completely elucidated. OPs are

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Table 1
Laboratory data.

Variable	Normal range	Result
Glucose	74–118 mg/dL	145
Sodium	136–144 mEg/L	131
Potassium	3.6–5.1 mEg/L	3.5
CK-MB	0–5.6 ng/mL	10.04
Troponin I	0–0.034 ng/mL	0.72
Myoglobin	0–43.3 ug/L	508
Hemoglobin	13.1–17.2 g/dL	12.6
Hemotocrit	39–50%	37.9
Leukocyte	4.5–10.5 per mm^3	16.6

powerful inhibitors of cholinesterases. As a consequence, they cause a massive accumulation of acetylcholine.[5] Although the most evident symptoms of acute organophosphate intoxication are related to increase of parasympathetic activity (bradycardia, hypoperfusion of myocardium), some signs of sympathetic overstimulation (tachycardia and systemic arterial hypertension) can also be observed in the heart.[6] But there was neither bradycardia nor tachycardia in our patient. His blood pressure was 110/70 mm Hg and his heart rate was 80 beats/min at admission time and no changes were observed during hospitalization period. On the other hand, clinical symptoms of our patient including vomiting, increased salivation and diaphoresis were fit as organophosphate poisoning symptoms and might due to the accumulation of acetylcholine. In the pathogenesis of myocardial infarction, coronary vasospasm is an important factor. It has been shown that in patients with atheromatous coronary artery like our patient, acetycholine caused coronary vasoconstriction even in low doses, paradoxically.[7]

Exposure to OPs compounds may increase the vulnerability of patients to stress and intensive exercise and may result in sudden death from myocardial infarction. Furthermore, Allon and et al. showed that an exposure to a potent acetylcholine esterase inhibitor (sarin vapor) in rats resulted in an increased sensivity to epinephrine and vulnerability to develop cardiac abnormalities.[5] This increased sensivity may explain the mortality observed following OPs exposure, especially under challenging conditions such as stress or intensive physical exercise as seen in our farmer patient.

Moreover, induction of stress, one of the characteristics of pesticides, is a response to every situations which consequently leads to enhanced release of catecholamines and other enzymes including histamine, neutral proteases (tryptase, chymase). These vasoactive amines penetrate the collagen matrix of the plaque, and produce erosions and rupture. Since it is known that atherosclerotic coronary artery has an increased sensivity to these vasoconstrictor agents,[8] all these mediators can cause myocardial injury and increase vulnerability of plaque which was existed in right coronary artery of our patient. In addition, the fact that leucocytosis is one of the laboratory findings of our patient suggest the existence inflammatory situation which may trigger the stress response. A rare manifestation of such reactions are referred to as Kounis syndrome.[9] Type II Kounis syndrome occurs in patients with atheromatous coronary disease whereby the hypersensivity reaction induces plaque erosion and rupture leading to the clinical manifestation of acute myocardial reaction.[10] Our patient was a possible variant of type II Kounis syndrome with preexisting coronary risk factors such as smoking, male gender, $>$ 45 years of age, atheromatous coronary artery. Therefore, the myocardial damage may be attributed to the direct cardiotoxic effects of OPs.

Present case manifested typical signs and symptoms of myocardial infarction and history of OPs poisoning together. So, it is possible to suggest that OPs intoxication may cause myocardial injury. OPs induced myocardial infarction seems to be mediated via direct toxic effect of compound in this case but further studies are needed to clarify the exact mechanism.

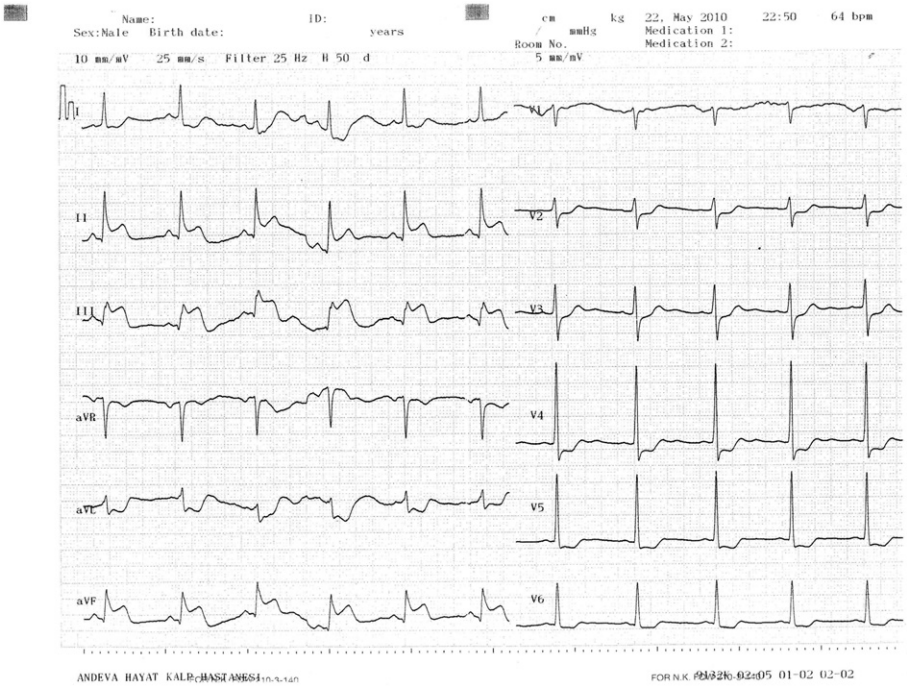

Fig. 1. The electrocardiogram showed 1–2 mm ST-segment elevation in DII, DIII and AVF derivations accompanied by 1–2 mm ST horizontal depression in DI-AVL leads.

Conflict of interest
None declared.

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None.

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